treatment with prednisolone ($p = 0.04$) was significantly higher than in patients with recurrent lupus enteritis.

The wall thickness of the most thickened bowel in patients with recurrent lupus enteritis (mean (SD); 9.0 (3.7) mm) was greater than in patients with non-recurrent lupus enteritis (6.1 (2.6) mm), although the difference was not significant ($p = 0.08$). All the five patients with bowel wall thickness ⩾9 mm experienced recurrence. This finding may be considered a high-risk factor for recurrent lupus enteritis.

Cumulative dose and duration of treatment may be influenced by other complications of SLE, including lupus nephritis. In our study, class II lupus nephritis was newly diagnosed during prednisolone tapering in three patients (2/7 in non-recurrent, 1/9 in recurrent group), prolonging the duration of treatment with prednisolone. Despite this confounding variable, the dose and duration of corticosteroids was related more closely to the recurrence of lupus enteritis than laboratory indices, including autoantibody profiles and SLEDAI score reflecting lupus activity.

Successful treatment with high-dose prednisolone and intravenous pulse cyclophosphamide has been reported in many patients with intestinal vasculitis, and immunosuppressants such as cyclophosphamide may be recommended for patients with severe or steroid-resistant lupus enteritis.[4 5] In conclusion, longer treatment with corticosteroids or treatment with additional immunosuppressants may be considered in patients with lupus enteritis and with initial bowel wall thickening >9 mm.

Authors' affiliations
Y G Kim, S S Nah, C-K Lee, H-B Moon, B Yoo, Division of Allergy and Rheumatology, Department of Internal Medicine, University of Ulsan College of Medicine, Asan Medical Center, Seoul, Korea
H K Ha, Division of Allergy and Rheumatology, Department of Radiology, University of Ulsan College of Medicine

Competing interests: None declared.

Correspondence to: B Yoo, Division of Allergy and Rheumatology, Department of Internal Medicine, University of Ulsan College of Medicine, Asan Medical Center, 388-1 Pungnap-dong, Songpa-gu, Seoul 138-736, Korea; byoo@amc.seoul.kr

Accepted 13 February 2006

REFERENCES

1 **Lee CK**, Ahn MS, Lee EY, Shin JH, Cho YS, Ha HK, *et al.* Acute abdominal pain in systemic lupus erythematosus: focus on lupus enteritis (gastrointestinal vasculitis). *Ann Rheum Dis* 2002;**61**:547–50.
2 **Ha HK**, Lee SH, Rha SE, Kim JH, Byun JY, Lim HK, *et al.* Radiologic reatures of vasculitis involving the gastrointestinal tract. *Radiographics* 2000;**20**:779–94.
3 **Byun JY**, Ha HK, Yu SY, Min JK, Park SH, Kim HY, *et al.* CT features of systemic lupus erythematosus in patients with acute abdominal pain: emphasis on ischemic bowel disease. *Radiology* 1999;**211**:203–9.
4 **Cabrera GE**, Scopelitis E, Cuellar ML, Silveira LH, Mena H, Espinoza LR. Pneumatosis cystoides intestinalis in systemic lupus erythematosus with intestinal vasculitis: treatment with high dose prednisone. *Clin Rheumatol* 1994;**13**:312–16.
5 **Grimbacher B**, Huber M, von Kempis J, Kalden P, Uhl M, Kohler G, *et al.* Successful treatment of gastrointestinal vasculitis due to systemic lupus erythematosus with intravenous pulse cyclophosphamide: a clinical case report and review of the literature. *Br J Rheumatol* 1998;**37**:1023–8.

Lack of association between signaling lymphocytic activation molecule family member 1 gene and rheumatoid arthritis in the French and Tunisian populations

G Chabchoub, E Petit-Teixeira, A Maalej, C Pierlot, Z Bahloul, F Cornélis, H Ayadi

Ann Rheum Dis 2006;**65**:1538–1539. doi: 10.1136/ard.2006.052209

Signaling lymphocytic activation molecule family member 1 (*SLAMF1*) is a member of the CD2 immunoglobulin superfamily, which is expressed in T cells, B cells, macrophages and dendritic cells.[1] *SLAMF1* is associated with B cell proliferation and immunoglobulin synthesis.[2] The expression of *SLAMF1* in peripheral blood B cells from patients with rheumatoid arthritis was raised compared with healthy controls,[3] and high expression of this molecule was observed in synovial fluid and tissue of patients with rheumatoid arthritis. Moreover, mice deficient in the *SH2D1A* molecule, which binds to the *SLAMF1* receptor, were susceptible to experimental autoimmune diseases.[4 5] Considering these biological data and the potential role of the *SLAMF1* gene in regulating the immune system, we investigated the candidate gene *SLAMF1* polymorphism, located on chromosome 1 (1q21.3–32.3), in patients with rheumatoid arthritis.

Three *SLAMF1* single-nucleotide polymorphisms were studied: C>A (rs 2295612) located in exon 1, G>C (rs 1809963) located in intron 4 and A>C (rs 3796504) located in exon 7. Firstly, 100 French patients with rheumatoid arthritis (87 women and 13 men; mean age: 32 years) in both parents were studied. Secondly, 103 Tunisian patients (85 women and 18 men; mean age: 50.7 years) and 111 healthy controls (64 women and 47 men; mean age: 54.8 years) were analysed in a case–control study. All patients selected fulfilled the 1987 American College of Rheumatology criteria for rheumatoid arthritis.[6]

The polymerase chain reaction-restriction fragment length polymorphism technique was used for genotyping all samples (*DdeI* for rs2295612; *BslI* for rs1809963; *Hpy188II* for rs3796504; primer sequences and amplification programmes given on request).

Statistical analysis was examined by three different methods: transmission disequilibrium test, genotype relative risk and affected family-based controls. Results from Tunisian control subjects and patients with rheumatoid arthritis were compared using the χ^2 test and Fisher's exact test. *SLAMF1* haplotypes in the French Caucasian trio and Tunisian population were inferred using Genehunter and Phase 2.1 softwares, respectively.

Allele frequencies for the three single-nucleotide polymorphisms were in accordance with Hardy–Weinberg equilibrium in both Tunisian controls and French family-based controls. Statistical analysis did not show any linkage disequilibrium between these markers in the Tunisian as well as in the French population ($p>0.05$). Transmission disequilibrium test analysis showed that rs2295612, rs1809963 and rs3796504 are not preferentially transmitted from heterozygotic parents to affected offsprings in the French trios with rheumatoid arthritis. Similar results were

Table 1 Analysis of the signalling lymphocytic activation molecule family member 1 single-nucleotide polymorphisms by affected family-based control, transmission disequilibrium and genotype relative risk tests in 100 French families with rheumatoid arthritis

	Allele frequencies			TDT			GRR
SNP alleles	Frequency transmitted	Frequency untransmitted	p Value	Transmitted	Untransmitted	p Value	p Value
rs 2295612_C	0.79	0.83	0.30	28	36	0.31	0.31
rs 2295612_A	0.21	0.17		36	28	0.31	
rs 1809963_C	0.99	0.995	0.31	1	3	0.31	0.62
rs 1809963_G	0.01	0.005		3	1	0.31	
rs 3796504_C	0.89	0.9	0.74	17	19	0.73	0.08
rs 3796504_A	0.11	0.1		19	17	0.73	

GRR, genotype relative risk; SNP, single-nucleotide polymorphism; TDT, transmission disequilibrium test.

obtained using genotype relative risk and affected family-based control tests (table 1).

Moreover, our results showed the same distribution of *SLAMF1* alleles and genotypes between Tunisian patients and controls ($p>0.05$).

Three different haplotypes (*CCC*, *CCA*, *ACC*) were identified at the *SLAMF1* gene, with a frequency >5% in the French and Tunisian populations. Neither a significant disequilibrium of transmission to patients with rheumatoid arthritis nor a significant difference in distribution of these haplotypes between Tunisian patients with rheumatoid arthritis and controls was found.

Our study is the first report to examine the potential role of the *SLAMF1* gene in the susceptibility to rheumatoid arthritis in both the French and Tunisian populations, which indicates that *SLAMF1* polymorphisms do not contribute to susceptibility to rheumatoid arthritis in the studied populations. Our study should be replicated in other populations to confirm our preliminary results. Genotypes are available at http://www.GenHotel.com.

ACKNOWLEDGEMENTS

We sincerely appreciate the invaluable comments and encouragement of Professor Nadir Farid for his critical reading of the manuscript.

Authors' affiliations

G Chabchoub, A Maalej, H Ayadi, Laboratoire de Génétique Moléculaire Humaine, Faculté de Médecine de Sfax, Rue Magida Boulila, Tunisia
E Petit-Teixeira, C Pierlot, F Cornélis, Genhotel Laboratoire de Recherche Européen pour la Polyarthrite Rhumatoïde, Université d'Evry-Val d'Essonne, Paris, France
F Cornélis, Unité de Génétique Clinique. Hôpital Lariboisière, Assistance publique, Hôpitaux de Paris, Paris
Z Bahloul, Service de Médecine Interne CHU Hédi Chaker Sfax, Sfax, Tunisia

Funding: AFP, ARP, Association Française des Polyarthritiques, Association de Recherche pour la Polyarthrite, Association Polyarctique, SFR, Genopole (France), Ministère de l'Enseignement Supérieur, Ministère de la Recherche Scientifique et de la Technologie (Tunisa) and the International Centre for Genetic Engineering and Biotechnology (ICGEB), Trieste (Italy).

Competing interests: None declared.

Correspondence to: H Ayadi, Laboratoire de Génétique Moléculaire Humaine, Faculté de Médecine, Avenue Majida Boulila 3029 Sfax, Tunisia; hammadi.ayadi@fmsf.rnu.tn

Accepted 29 January 2006

REFERENCES

1 **Veillette A**, Lattour S, Davidson D. Negative regulation of immunoreceptor signalling. *Annu Rev Immunol* 2002;**20**:669–707.
2 **Punnonen J**, Cooks BG, Carballido JM, Bennett B, Peterson D, Aversa G, *et al*. Soluble and membrane-bound forms of signaling lymphocytic activation molecule (*SLAM*) induce proliferation and Ig synthesis by activated human B lymphocytes. *J Exp Med* 1997;**185**:993–1004.
3 **Isomaki P**, Aversa G, Cooks BG, Luukkainen R, Saario R, Toivanen P, *et al*. Increased expression of signalling lymphocytic activation molecule with rheumatoid arthritis and its role in the regulation of cytokine production in rheumatoid synovium. *J Immunol* 1997;**159**:2986–93.
4 **Hron JD**, Caplan L, Gerth AJ, Schwartzberg PL, Peng SL. *SH2D1A* regulates T-dependent humoral autoimmunity. *J Exp Med* 2004;**200**:261–6.
5 **Komori H**, Furukawa H, Mori S, Ito MR, Terada M, Zhang MC, *et al*. A signal adaptor SLAM-associated protein regulates spontaneous autoimmunity and Fas-dependent lymphoproliferation in MRL-Faslpr lupus mice. *J Immunol* 2006;**176**:395–400.
6 **Arnett FC**, Edworthy SM, Bloch DA, McShane DJ, Fries JF, Cooper NS, *et al*. The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. *Arthritis Rheum* 1988;**31**:315–24.

Palindromic rheumatism: different genetic background implies a distinct disease entity

S-K Kim, H-S Lee, K W Lee, S-C Bae, J-B Jun

Ann Rheum Dis 2006;**65**:1539–1540. doi: 10.1136/ard.2006.052928

Palindromic rheumatism is characterised by multiple, episodic and recurrent attacks of arthritis, accompanied by periarticular inflammation without residual joint damage, occurring at irregular intervals and lasting from a few hours to several days.[1–3]

The definite pathogenesis and disease entity of palindromic rheumatism has not been clarified until now. We investigated the association of rheumatoid arthritis and palindromic

Abbreviation: HLA, human leucocyte antigen